Williams (J. W.)
National Board of Health.
Compliments of The author.

DIPHTHERIA.

READ BEFORE THE

RICHMOND ACADEMY OF MEDICINE AND SURGERY,

FEBRUARY 16TH, 1892,

BY DR J. W. WILLIAMS.

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DIPHTHERIA.[1]

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THE human body is covered by skin; and the disgestive tract, urethra, vagina, &c., by mucous membrane. Both skin and mucous membrane are covered by a layer of epithelium that is so arranged that solution of continuity must take place before bacteria or germs can have access to the tissues within. Hence, a wound of this epithelium is absolutely necessary to infection—to sepsis. In other words, septicæmia or pyæmia cannot take place without a wound in this epithelial layer, through which wound the bacteria enter the blood and systemic intoxication follows as a consequence. In case III[2] below, Mr. C. had a minute scratch near the nostril, so small as to escape my notice, and in using a towel that I had used in opening an abscess upon his brother's face, who was suffering from erysipelas, he inoculated himself with the same disease with which his brother was suffering. The streptococci entered the blood through this microscopic wound, and septic intoxication followed. Case IV affords another example. Mr. R. was suffering from pyæmia. The streptococcus was found in the pus from an abscess near the outer middle of right thigh. From this abscess the streptococci entered the general circulation through the disintegrated or broken walls of the neighboring blood-vessels, and pyæmia resulted.

A few words upon the process of

INFLAMMATION

involved in diphtheria may not be out of place. All injuries causing wounds, whether the keen edge of the flashing stiletto or the jagged and rough section of a shell, destroy the life of those cells that lie in the path of the cutting or lacerating object. The blood and lymph exuding from

1. The old Roman physicians called it Angina, and the Greeks Cynanche—"Nostri *anginam* vocant: Graecos nomen *cynauche*."—Celsus, caput IV, p. 257. Its present name (*Diphtheria*) was given by M. Bretennou, of France in 1818. It is thought that descriptions of this disease can be traced to a time anterior to Hippocrates (460 B. C.), and that Aretanes, of Cappadocia, described it (100 A. D.). One of the earliest treatises was by Hecker, who described an epidemic that prevailed in Holland in 1337. Dr. Fothergill gives a description of the epidemic that appeared in London in 1745, and Dr. Samuel Bard, of New York, describes the epidemic that swept this country in 1771.

2. For cases III, IV, see paper on Pyogenesis.

the vessels coagulate, and necrosis follows. If a number of active micrococci from the surrounding air or dirt attack this wound, they find a congenial soil for their development and multiplication. The fermentative decomposition here set up produces certain chemical alkaloids or extremely poisonous substances—the *ptomaines.* The adjacent vaso-motor nerves come under their toxic influence, causing active hyperæmia, and we have the first symptom of inflammation—"*rubor.*" The dilated capillaries involved, in virtue of this very dilatation, invite a larger volume of blood, which now rushing through them with greater volocity, the red blood-corpuscles become packed and finally stagnate (stasis) in the smaller arteries. The walls of these vessels give way, and the white blood-corpuscles and serum are extravasated into the surrounding tissues, infiltrating their interstices, and the characteristic *swelling* ensues ("*turgor*"), the second symptom of inflammation. As a consequence of increased blood supply, marked oxydation takes place, a rise of the local temperature follows, and we have "*calor*" (heat), the third symptom of the inflammatory action. The action, perhaps, of the ptomaines, the direct pressure caused by the swelling, combined with the actual destruction of the nerve tissue during the resulting suppuration, accounts for the *pain* ("*dolor*") which completes the classical cycle of the four cardinal symptoms of inflammation—*rubor, calor, turgor, dolor.*

The infiltrated tissue, devitalized by the shutting off of the normal circulation, falls a prey to the millions of micro-organisms, and necrosis logically follows. This last step in the textual destruction results in the liquefaction of the tissues. A cavity or abscess is formed, filled with lymph-serum exuded from the injured blood-vessels in the neighborhood, millions of dead white blood-corpuscles (pus cells), and shreds of necrosed tissue. The great tension surrounding a large abscess necessarily causes an overflow of its liquid contents into the surrounding efferent vessels—veins and lymphatics. The ptomaines at once enter the general circulation, systemic intoxication ensues, manifested by a marked rise of temperature, rigors, nausea, headache, delirium, and asthenia—septic fever. This systemic intoxication is caused, not by the absorption of the bacilli, but its ptomaines—a very poisonous substance, a white powder, soluble in water, whose chemistry is $C_{45.35}$, $H_{7.13}$, $N_{16.33}$, $S_{1.39}$, $O_{29.80}$.—(Vaughan.) The extension of septic material is twofold: First, by infiltrating the tissue interstices by columns of micrococci; and secondly, by way of the lymphatics. It seems probable that in diphtheria the Klebs-Hoeffler bacillus utilize both of these ways. If the parts affected are loose tissues, as the tonsils and cervical glands, the infection will be rapid; if the parts are dense, the inflammation is localized so long as the tissue-density resists the pressure of the imprisoned secretions. Inflammation has three stages:

First stage. Dilatation of the blood-vessels, the increased flow of blood

and lymph to the part, slowing of the circulation, and finally inflammatory stasis, with extravasation of blood-plasma and corpuscles. In this stage the vital activity of the tissue is suspended, the functions of the part are, so to speak, paralyzed; in other words, the tissue has, to a certain extent, become devitalized, and therefore unable to resist the entrance of bacteria.

Second stage. Here this weak tissue has been removed and healthy granulations have taken its place. This healthy granulation seems to have power to resist attacking organisms.

Third stage. Here the irritating agent no longer acts. The first effect of this irritant was to damage the part, and under its damaging influence dilatation of the blood-vessels resulted; and as a consequence of this dilatation an increased flow of blood and lymph took place. Nature is here trying to overcome the irritating cause by flushing the part with blood—by washing away the obstruction. Probably this process is often going on in our bodies unnoticed by us. If the irritant continues to act, and these measures are ineffectual, nature removes the irritating cause by necrosis and sloughing.

The tonsils and cervical glands being vascular bodies, and loose in their anatomical structure, they become easily infiltrated by columns of micrococci, which burrow down to the depth of a *quarter of an inch* in the tissue and *below* the surface (Prudden-Seibert). Here inflammation takes place, which results in an exudation of serum and leucocytes accumulating in the upper epithelial layer of the mucosa, thus producing the false membrane of diphtheria. In view of this pathological fact, all washes and gargles are useless, just because they do not and cannot reach down to the microbe burrowing beneath the surface. The mortality from diphtheria in the city of New York during ten months of the year 1890 amounted to 1,725 out of 4,340 cases, and nowhere is the bichloride treatment more extensively used than in New York; yet here is frightful mortality. "Of the strength of 1-500, thousands of micrococci remained alive at the end of fifteen minutes."—(Pruden.) Given of this strength you kill your patient, and in weak solutions you fail to kill the streptococcus; and in either case you fail to reach the microbe. This treatment is not efficient, because it does not reach the bacilli at work below the mucosa. It does not penetrate the false membrane, but glides off its surface into the œsophagus; it is not local, because the seat of the disease is not reached; and it is not germicidal, because the solution is not strong enough to destroy the diphtheritic germs. Chlorate of potassa, benzoate of soda, boric acid, *et omne genus*, may at once be struck from the list as topical agents useful in diphtheria. In the very nature of diphtheria the only scientific treatment must be a germicidal one. Carbolic acid and corrosive sublimate solutions, when used of sufficient strength, will inevitably produce systemic intoxication. The sooner local treatment is commenced the better the chances of preventing the general infection. An early diagnosis is therefore in-

dispensable. A bit of the pseudo-membrane should at once be removed, dried on a cover-glass, stained with fuschin, or gentian violet, and placed under the microscope for examination.[3] The microscope was invented about the latter part of the sixteenth century, and Kircher suggested in 1646 that disease might be due to minute organisms. Van Leuwenhorck, of Holland, pushed his investigations still further (1680–1723); Andy in 1701, Muller in 1786, Ehrenburg in 1833. But it was not until 1863 that Davani established a connection between bacteria and disease. And the first complete study of a contagious affection was made by Pasteur in 1869; then Koch in 1875; and finally Klebs-Loeffler differentiated the bacteria of diphtheria in 1884: "Small, slightly-curved rods, about as long as the tubercle bacilli and twice as broad; the ends are at times swollen." Dr. Plfeifer discovered the bacillus of *la grippe* in 1891.

INOCULATION.

Breiger and Frankel, by injecting ten to twenty per cent. of a three-weeks'-old culture of diphtheria bacilli, produced an immunity in guinea-pigs against the virulent form. Drs. Wood and Formad, of the University of Pennsylvania, have been invited by the National Board of Health "to determine whether it is possible to produce diphtheria in the lower animals by inoculation." Seibert has inoculated eighty animals. Here, stretching out before us, is a new and most inviting field for the scientific physician. The day will come soon and shortly when children[4] will be inoculated to insure immunity against diphtheria as well as from small-pox. Where is the Jenner who will immortalize himself and hand down to posterity a name more precious than a monument of diamonds?

TREATMENT.

Submembraneous and Tonic.—Diphtheria is a local disease, and the constitutional symptoms are due to the absorption of the ptomaines from the local lesion. The treatment is essentially germicidal and tonic. Both the pathology and etiology of the disease teach this. Besides, it is the most scientific and successful. "It should be clearly held in mind—by those eager to draw from experimental studies on the etiology of this disease such practical lessons as shall be of value in treatment—that it seems to be fully established that in all of the cases the seat of infection and the origin of the

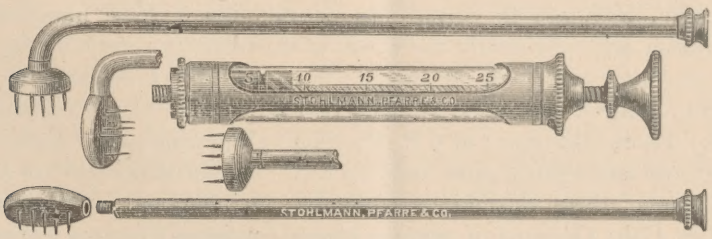
3. "There are only two germs to be considered in studying the etiology of diphtheria—the *streptococcus* and the *rod-shaped bacillus* with rounded extremities."—(Oertel.) After all that has been written upon this qestion, I think it is conservative to say that the identification of the specific microbe and its ptomaines will be the work of the future.

4. The important discovery made by Behring and Kitasato in 1890, that blood-serum taken from animals that had been rendered immune to tetanus and to diphtheria could cure other animals affected with these diseases, and the further discovery made by the Drs. Klemperer, that serum taken from an animal that had pneumonia contains the *anti-pneumotoxin*, by means of which an immunity against pneumonic septicæmia is secured in other animals, should stimulate my medical brethren to push their investigations in this new field under a brighter light—encouraged by a stronger hope.

mischief is *always* a *local* one."—(Prudden's "Studies on the Etiology of Diphtheria.")

The pseudo-membrane is an exudate from the deeper layer of the mucous membrane, coagulated in the epithelium, and not the disease, but the result of it. If it is removed, mopped, or torn, the points of infection will be multiplied. Let it alone. Geppert has shown that "bacilli will live in a 7 per cent. solution of carbolic acid, and in a 1-1000 solution of the bichloride of mercury for twenty minutes." He also showed that the anthrax bacillus died in ten seconds if brought in contact with a 0.2 per cent. solution of aqua chlori. "The obvious lesson taught by a definite conception of the nature of the germ which causes diphtheria is not to dally with fancy mixtures, which have at least a moderate germicidal power, but to get at the growing germ, as directly as the seat of the lesion will permit, with some agent that we know will kill it."—(Prudden).

Traube first made intratonsular injections in diphtheria, and after him Huebner. Traube used a 3 to 5 per cent. solution of carbolic acid twice daily, and reduced the mortality from 35.5 per cent. to 10 per cent. Seibert, in 1891, treated thirty-five cases hypodermically, and only lost two. I treated lately eleven cases, with one death. Case six recovered and was out on the streets for eight or ten days, but was stricken with paralysis, and died on the forty-second day. The hypodermic syringe I use has five tubes or needles fixed to a plate, which is screwed to the barrel, making the instrument eight inches long. These five needles are one-fourth of an inch in length. The plate is firmly held against the tonsil or pharynx, the needles boldly pressed through the false membrane down to the submucous tissue where the bacilli are at their deadly work, and the aqua chlori at once, by injection, brought in contact with them. In the mean time, however mild the case, I at once put the patient upon tonics:



℞. Best beef . . . . . 1 lb.
Aqua . . . . . Oij.
Boil down to one pint, strain, and season with salt and pepper, and take daily.

Should the constitutional symptoms announce the general systemic infection, the patient being stronger, will be the more readily tided over it.

Case I. Mr. R., age 26, October 2d, 7 P. M.: Fever 104° for two days previous; false-membrane on both tonsils and infiltration of cervical glands.

Injection of 30 gtt. of aqua chlori at 7:15 P. M. into both tonsils through the pseudo-membrane down to the depth of a quarter of an inch. October 3d, 9 A. M.: Œdæma of both tonsils continue. Intense redness of throat; voice changed. Injection at 9:15 A. M.; injection again the next day. False membrane becoming detached. October 7th, discharged cured. Beaf tea, one pint daily, was given this case.

Case II. Mr. C., age 18 years, October 9th: Fever high; false membrane on right tonsil, smaller patch on left; œdæma and infiltration of cervical glands. Injections. October 10th, 9 A. M.: Patient worse; œdæma increasing, especially of the left cervical glands. Seven P. M.: Injection, each time into both tonsils. October 11th: No fever; false membrane an inch long; patient worse. Nine A. M.: Injectin; antiseptic gargle to clear throat; injections daily; beaf-tea (1 lb.). October 12th, discharged cured.

Case III. Mr. C., age 21: High fever; false membrane on both tonsils; injection in both tonsils as before; liver torpid.

℞. Euonymin . . . . . grs. iv
Leptandrin . . . . . grs. iii
Podophyllin (neutral) . . . . . grs. ij
M. Sig. Ft. pil. ix. Dosis: Unus omni tri hora.

This corrected all hepatic trouble, cleaned off the tongue, and cleared up the complexion. First saw this man November 24th. Made daily injections for three days. November 27th: Membrane came away. Discharged cured.

Case IV. Boy, age 10 years. High fever; false membrane on right tonsil, and on second day on left tonsil also. Injection through false membrane into both tonsils to the depth of a quarter of an inch. On third day false membrane came off. Discharged cured.

Case V. Boy, age 4½ years. (In consultation.) False membrane on both tonsils and over nearly the whole of the pharynx; systemic intoxication; the septicæmia well developed; heart feeble. First saw him October 15th. Injection at 11 A. M.; injection at 6 P. M. October 16th, 10:30 A. M.: Three injections; at 6 P. M. coughed up tube cast. False membrane loose and a piece came away an inch long and one eighth of an inch in thickness. Sent this fine specimen to Dr. Billings, at Washington. October 17th: Œdæma of cervical glands continues; croupy symptoms; bronchi involved; pulse failing.

℞. Peroxid. of hydrogen . . . . . ℥ij.
Tr. digitalis . . . . . ℥i
Fl. ext. sanguinaria cand. . . . . ℥ij
Dosis: Coch. parv. omni tri hora.

Septicæmia no doubt caused by absorption of the ptomaines. Beef tea. Alimentary tract disinfected with thymol. Patient gradually growing

worse. October 18th, 2 P. M.: Died of septic infection. I do not think any local treatment will avail after systemic septic intoxication sets in.

CASE VI. Boy, age 3½ years; Tonsils covered with false membrane; five or six ulcers on leg; had been exposed to the disease; false membrane covered each ulcer. This boy recovered; was out on the streets for a week or so; fattened rapidly, but died on the forty-second day of paralysis,[5] as mentioned above.

CASE VII. Lady, age 26; false membrane on both tonsils; injection at once through the necrosed membrane down to the mucosa beneath where the microbes are burrowing; injections daily; discharged cured on the fifth day.

HISTOLOGY.

Prudden found the streptococcus pyogenes in twenty-two out of twenty-four cases of diphtheria examined by him. We have reasons for believing, on biological and experimental grounds, that not only is the streptococcus pyogenes the etiological factor in diphtheria, but that erysipelas and some forms of phlegmonous inflammations are cognate[6] diseases with diphtheria. Pruden carried cultures of streptococci from cases of diphtheria along side by side with cultures of streptococci made from various cases of acute erysipelas and phlegmonous inflammation, week after week; over and over again has he measured and compared the growth from these three sources. He has repeatedly inoculated duplicate sets of animals with the different cultures, and has never found a single, constant feature of difference between them. The close relationship existing between these three forms of inflammation has long since been pointed out by Baumgarten.

In the above cases I had the bedding, napkins, towels, &c., boiled in water for two hours in the same rooms in which I treated the cases. The city should establish a disinfecting-house,[7] well equipped, for this purpose, to which all clothing, bedding, &c., from infectious diseases should be sent. All exudates should be received into vessels containing a 5 per cent. solution of carbolic acid.

The following mortuary table shows the alarming mortality of the city of Richmond—a death-rate of over 44 per cent. It will be seen that of the forty-one leading cities of the United States Richmond exceeds them all probably in her death-rate—125 deaths out of 279 cases of diphtheria:

5. "Paralysis occurs in 40 per cent. of the cases."—*Brower.*

6. The uniform presence of the streptococcus in these three diseases, as an etiological factor, indicates a closer relationship than had been supposed, and suggests that future biological studies may demonstrate their identity.

7. The sulphur fumigation of infectious houses is under the ban of a more advanced science. A large and well-arranged disinfecting plant is now being planned for the city of New York.

MONTHLY RETURNS OF MORTALITY

—OF—

Forty-two Cities of the United States.

PLACES AND POPULATION.		Diphtheria and Croup.
New York.	Jan.	156
	Feb.	185
	Mar.	194
1,687,072.	Apr.	166
	May	131
	Jun.	333
	July.	134
	Aug.	111
	Sep.	139
	Oct.	176
Chicago.	Jan.	133
	Feb.	117
	Mar.	118
1,200,000.	Apr.	75
	May.	88
	Jun.	69
	July.	58
	Aug.	93
	Sep.	107
	Oct.	151
Philadelphia.	4 Jan.	86
	Feb.	83
	4 Mar.	79
1,069,264.	5 Apr.	91
	4 May.	71
	4 Jun.	54
	5 July.	54
	4 Aug.	52
	5 Sep.	94
	4 Oct.	188
Brooklyn.	4 Jan.	130
	Feb.	102
	4 Mar.	90
871,723.	5 Apl.	121
	4 May.	93
	4 Jun.	81
	5 July.	78
	4 Aug.	47
	5 Sep.	82
	4 Oct.	96
St. Louis.	Jan.	30
	Feb.	30
	Mar.	29
460,000.	Apr.	29
	May.	26
	Jun.	26
	July.	19
	Aug.	16
	Sep.	
	Oct.	

PLACES AND POPULATION.		Diphtheria and Croup.
Baltimore.	Jan.	30
	Feb.	31
	Mar.	23
455,427.	Apr.	14
	May.	28
	Jun.	24
	July.	14
	Aug.	18
	Sep.	35
	Oct.	49
Boston.	Jan.	22
	Feb.	21
	Mar.	18
459,062.	Apr.	
	May.	30
	Jun.	17
	July.	7
	Aug.	10
	Sep.	12
	Oct.	33
Cincinnati.	Jan.	34
	Feb.	26
	Mar.	17
300,000.	Apr.	16
	May.	20
	Jun.	15
	July.	26
	Aug.	18
	Sep.	17
	Oct.	29
San Francisco.	Jan.	45
	Feb.	61
	Mar.	63
330,000.	Apr.	39
	May.	26
	Jun.	28
	July.	17
	Aug.	19
	Sep.	24
	Oct.	37
New Haven, Conn.	Jan.	5
	Feb.	7
	Mar.	7
	Apr.	1
86,000.	May.	9
	Jun.	6
	July.	1
	Aug.	5
	Sep.	
	Oct.	

PLACES AND POPULATION.		Diphtheria and Croup.
Buffalo.	Jan.	42
	Feb.	25
	Mar.	13
255,664.	Apr.	19
	May.	19
	Jun.	13
	July.	13
	Aug.	16
	Sep.	26
	Oct.	39
New Orleans.	Jan.	4
	Feb.	3
	Mar.	4
254,000.	Apr.	10
	May.	2
	Jun.	1
	July.	2
	Aug.	1
	Sep.	3
	Oct.	17
Wash-ington.	Jan.	24
	5 Feb.	16
	4 Mar.	8
	Apr.	13
250,000.	May.	10
	4 Jun.	13
	5 July.	21
	4 Aug.	16
	5 Sep.	34
	4 Oct.	34
Pittsburgh.	Jan.	19
	Feb.	30
	4 Mar.	17
247,000.	5 Apr.	12
	4 May.	11
	4 Jun.	14
	5 July.	20
	4 Aug.	24
	5 Sep.	49
	Oct.	
Louisville.	4 Jan.	15
	Feb.	9
	5 Mar.	7
227,000.	4 Apr.	11
	4 May.	1
	4 Jun.	8
	5 July.	7
	4 Aug.	7
	5 Sep.	9
	4 Oct.	41

MONTHLY RETURNS OF MORTALITY.

PLACES AND POPULATION.		Diphtheria and Croup.
Detroit. 220,000.	Jan.	41
	Feb.	27
	Mar.	48
	Apr.	24
	May.	31
	Jun.	15
	July.	16
	Aug.	19
	Sep.	27
	Oct.	34
Milwaukee. 220,000	Jan.	45
	Feb.	48
	Mar.	41
	Apr.	44
	May.	30
	Jun.	22
	July.	22
	Aug.	35
	Sep.	23
	Oct.	53
Minneapolis. 164,738.	Jan.	15
	Feb.	12
	Mar.	9
	Apr.	3
	May.	14
	Jun.	7
	July.	8
	Aug.	4
	Sep.	9
	Oct.	14
Jersey City. 67,237.	Jan.	22
	Feb.	18
	Mar.	14
	Apr.	14
	May.	10
	Jun.	31
	July,	23
	Aug.	17
	Sep.	
	Oct.	
St. Paul. 150,000.	Jan.	23
	Feb.	8
	Mar.	9
	Apr.	
	May.	4
	Jun.	3
	July.	
	Aug.	4
	Sep.	3
	Oct.	

PLACES AND POPULATION.		Diphtheria and Croup.
Rochest-r. 138,327	Jan.	11
	Feb.	6
	Mar.	
	Apr.	6
	May.	13
	Jun.	9
	July.	4
	Aug.	3
	Sep.	7
	Oct.	25
Providence, R. I. 135,000.	Jan.	5
	Feb.	6
	Mar.	9
	Apr.	
	May.	6
	Jun.	4
	July.	4
	Aug.	3
	Sep.	
	Oct.	11
Kansas City. 132,416.	Jan.	7
	Feb.	3
	Mar.	
	Apr.	
	May.	4
	Jun.	
	July.	
	Aug.	
	Sep.	3
	Oct.	7
Columbus. 101,945.	Jan.	12
	Feb.	9
	Mar.	8
	Apr.	3
	May.	6
	Jun.	5
	July.	5
	Aug.	4
	Sep.	3
	Oct.	11
Richmond, Va. 100,000. Total: Cases, 279. Deaths, 125.	Jan.	3
	Feb.	6
	Mar.	1
	Apr.	
	May.	3
	Jun.	3
	July.	38
	Aug.	48
	Sep.	79
	Oct.	75
	Nov.	15
	Dec.	8

PLACES AND POPULATION.		Diphtheria and Croup.
Albany. 100,000.	Jan.	6
	Feb.	11
	Mar.	8
	Apr.	1
	May.	1
	Jun.	5
	July.	3
	Aug.	1
	Sep.	5
	Oct.	
Syracuse, N. Y. 88,000.	Jan.	2
	Feb	3
	Mar.	2
	Apr.	2
	May.	
	Jun.	3
	July.	1
	Aug.	
	Sep.	3
	Oct.	
Toledo. 82,652.	Jan.	21
	Feb.	7
	Mar.	7
	Apr.	7
	May.	10
	Jun.	4
	July.	7
	Aug.	3
	Sep.	2
	Oct.	16
Scranton, Penn. 100,000.	Jan.	14
	Feb.	13
	Mar.	10
	Apr.	8
	May.	6
	Jun.	1
	July.	
	Aug.	3
	Sep.	4
	Oct.	5
Grand Rapids, Mich. 85,000.	Jan.	9
	Feb.	4
	Mar.	9
	Apr.	6
	May.	3
	Jun.	4
	July.	
	Aug.	11
	Sep.	12
	Oct.	12

MONTHLY RETURNS OF MORTALITY,

PLACES AND POPULATION.		Diphtheria and Croup.
Lowell, Mass. 80,400.	Jan.	2
	Feb.	1
	Mar.	
	Apr.	
	May.	4
	Jun.	1
	July.	1
	Aug.	1
	Sep.	5
	Oct.	
Paterson, N. J. 800,000.	Jan.	18
	Feb.	3
	Mar.	14
	Apr.	20
	May.	11
	Jun.	4
	July	19
	Aug.	5
	Sep.	24
	Oct.	15
Fall River, Mass. 75,000.	Jan.	5
	Feb.	2
	Mar	
	Apr.	1
	May.	1
	Jun.	
	July.	2
	Aug.	
	Sep.	
	Oct.	
Denver. 106,713.	Jan.	28
	Feb.	19
	Mar.	12
	Apr.	6
	May.	12
	Jun	26
	July.	14
	Aug.	14
	Sep.	
	Oct.	

PLACES AND POPULATION.		Diphtheria and Croup
Nashville, Tenn. 76,369.	Jan.	3
	Feb.	1
	Mar.	
	Apr	
	May.	1
	Jun.	1
	July.	
	Aug.	2
	Sep.	
	Oct.	3
Los Angeles, Cal. 65,000.	Jan	4
	Feb	2
	Mar.	1
	Apr.	3
	May.	2
	Jun.	2
	July	4
	Aug.	
	Sep.	1
	Oct.	
Troy, N. Y. 60,605.	Jan.	
	Feb.	2
	Mar.	3
	Apr.	3
	May.	1
	Jun.	4
	July.	2
	Aug	4
	Sep.	3
	Oct.	
Wilmington, Del. 62,000.	Jun.	2
	July.	9
	Aug.	6
	Sep.	10
	Oct.	

PLACES AND POPULATION.		Diphtheria and Croup.
Dayton, Ohio. 60,000.	Jan.	12
	Feb.	6
	Mar.	6
	Apr	7
	May.	5
	Jun.	7
	July.	5
	Aug.	6
	Sep.	1
	Oct.	5
Charleston, S. C. 65,145.	4 Jan.	
	Feb.	2
	Mar.	
	5 Apr	
	4 May	
	4 Jun.	
	5 July	
	4 Aug.	
	5 Sep.	
	4 Oct.	
Memphis, Tenn. 60,000.	Jan.	8
	Feb.	1
	Mar	2
	Apr.	
	May.	1
	Jun.	3
	July	3
	Aug.	8
	Sep.	7
	Oct.	1
Worcester, Mass. 84,655.	Aug.	11
	Sep.	4
	Oct.	
	Nov.	
	Dec	